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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE
DISEASES OF INFANTS AND CHILDREN
JOHN FITCH LANDON, M.D., Editor

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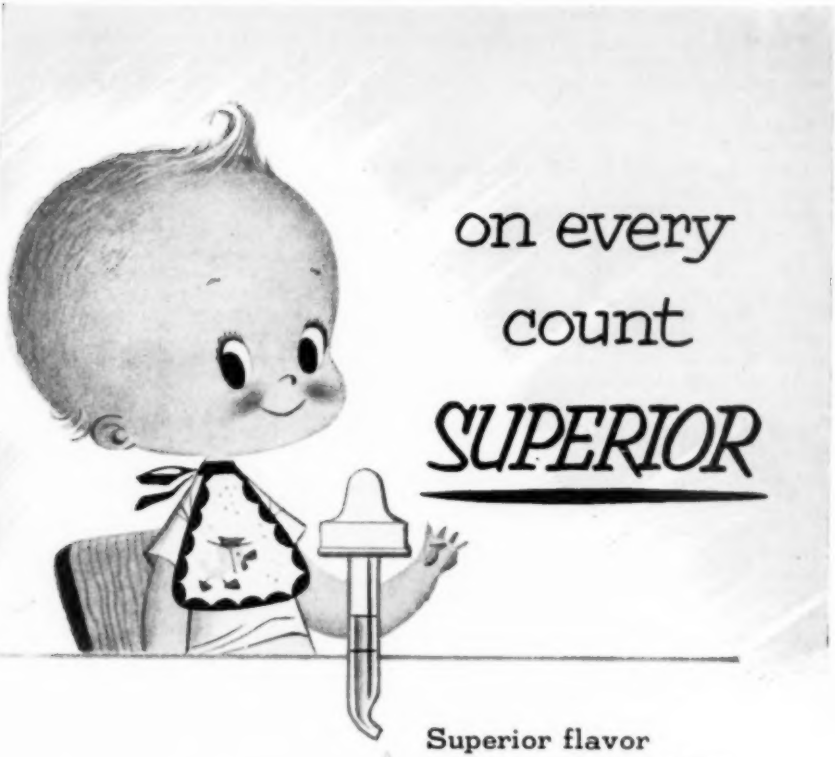

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In this latest study, each sample was analyzed separately. The analyses showed that MINUTE MAID Fresh-Frozen Orange Juice was equal to, or superior to, the home-squeezed juice in *all* of the components listed below:

TABLE

Mean Values in Samples Tested

COMPONENT	UNITS	MINUTE MAID FRESH-FROZEN ORANGE JUICE	HOME-SQUEEZED ORANGE JUICE
Betaine	mg./100 ml.	49	46
Biotin	mcg./100 ml.	0.26	0.26
Choline	mg./100 ml.	12	12
Cobalt	mcg./100 ml.	74	67
Folic acid	mcg./100 ml.	2.2	2.2
Iodine	mcg./100 ml.	0.24	0.21
Manganese	mcg./100 ml.	33	18
Nitrogen			
Total	mg./100 ml.	104	79
Amino	mg./100 ml.	22	22
Volatile	mg./100 ml.	8	7
Non-volatile	mg./100 ml.	96	72
Pantothenic acid	mcg./100 ml.	146	145
Para-amino-benzoic acid	mcg./100 ml.	4	4
Phosphorus	mg./100 ml.	19	18
Potassium	mg./100 ml.	380	290
Riboflavin	mcg./100 ml.	18	17
Tocopherols	mg./100 ml.	107	104
Vitamin A	mcg./100 ml.	19	16
Thiamine	mcg./100 ml.	87	83
Vitamin B_{12}	mcg./100 ml.	0.0022	0.0012

Although the results are again susceptible to variation according to crop and year, Fresh-Frozen MINUTE MAID was equal to the home-squeezed juice in the samples tested for the largest number of components listed; and in the mean values for iodine, manganese, potassium, Vitamins A and B_{12}, MINUTE MAID showed appreciably *higher* values.

SUMMARY

These new findings help enlarge professional knowledge of the nutrient constituents of orange juice in general and add fresh evidence that, on a cost basis, MINUTE MAID Fresh-Frozen Orange Juice has significant dietary advantages. Penny for penny, MINUTE MAID offers not only *more* Vitamin C, but also *more* of all the other vitamins and minerals listed than home-squeezed orange juice.

Taken in conjunction with the previously published findings, this should confirm the choice of physicians who recommend MINUTE MAID in place of home-squeezed orange juice.

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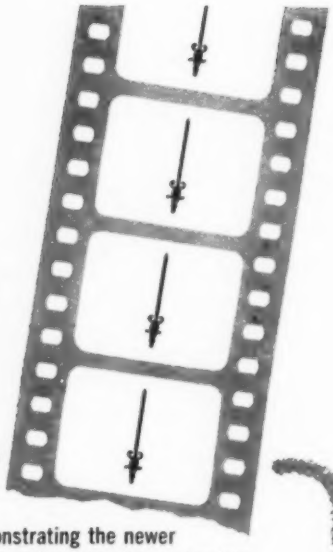

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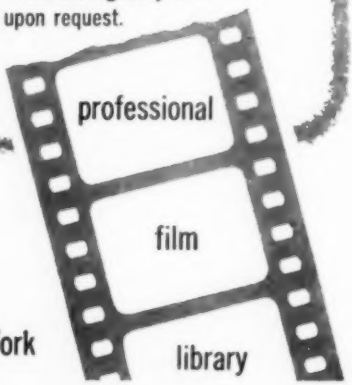

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THE EFFECT OF SUPPLEMENTARY dl-METHIONINE ON THE GROWTH OF FULL TERM INFANTS

LOUIS S. GOLDSTEIN, M.D.*

Yonkers, N. Y.

Methionine is an indispensable sulfur-containing amino acid which is known to play an important part in the prevention and treatment of hepatic and renal damage resulting from low protein and high fat diets and from various forms of intoxication. It has been found that the racemic form, namely dl-methionine, in suitable amounts, provides equivalent results to the biologically active levorotatory form.

The addition of dl-methionine to the diet of full term infants has been reported[1] to inhibit and remove excess ammonia in the urine and to prevent or improve napkin dermatitis, due specifically to ammoniacal urine. The present study was undertaken to measure the effect of supplementary dl-methionine upon growth in weight and to determine the maximum amount of dl-methionine which would not impair growth. Increase in length was not considered in the present study because it is well-known that, when growth during infancy is retarded, weight is more strikingly influenced than total body length[2].

The experiments of Beach and White[3] show that methionine stimulates the growth of animals previously stunted by a diet in which arachin (a globulin from peanuts), with its low methionine content, served as the sole protein.

Wretlind[4] fed dl-methionine to rats on a "synthetic" diet at

*Director of Pediatrics, Professional Hospital, Yonkers, N. Y.

levels ranging from 0.125 to 8.0 per cent for a period of ten days. Maximum growth was obtained on the 1 per cent level, with an average gain in weight of approximately 13.5 grams; on a diet containing 2 per cent methionine, the gain in weight was definitely less (approximately 7 grams); and at the 4 and 8 per cent levels, there was actually a loss in weight.

In another study[5], good growth in weaning rats was attained with diets containing 0.4 to 0.8 per cent of methionine, whereas 1.4 per cent in the diet impaired growth. Consequently, it has been demonstrated that in rats an increase in the methionine content of the diet of approximately 100 per cent may be detrimental.

PRESENT STUDY

In the present study, one group of infants receiving a proprietary milk formula designed especially for normal full term infants (control group) was compared with three (experimental) groups receiving an identical formula in addition to 25 per cent, 50 per cent and 100 per cent, respectively, dl-methionine supplement daily.

The study was conducted from September 1952 to June 1953. The effect of seasonal variation was reduced by pooling results over this period of time.

MATERIALS AND METHODS

One hundred and eighty-nine infants were included in this study. They were unselected private patients who were brought to the office for nutritional supervision.

1. *Constitutional Habitus.* In infants as well as adults, it is possible to recognize fundamentally opposite types of human beings (leptosome and pyknic). At one extreme we note the long, thin individual who has a delicate framework, weak and deficient musculature, long extremities, narrow face and narrow, dolichocephalic skull. At the other extreme we recognize the broad individual with short neck, barrel-shaped chest, round skull, short extremities; he has a strong, heavy, bony framework with good fat padding. The extremes, leptosome and pyknic, make up approximately 10 to 15 per cent at the opposite poles of the human form, and the intermediary zone is naturally made up of mixtures of every degree[6]. We also recognize the unusually small framed, short individual (ateleiosis) and the unusually large infant (macrosomia or gigantism).

Our experimental and control groups are sufficiently large to include a like number of divergent individuals for statistical analysis.

2. *Diet-Control Group.* The distribution as to diet was determined as follows: Breast fed infants were not included in the study. Infants who were not being breast fed were placed on the formula described below. The decision as to whether an infant was to be placed in the experimental group or the control group was based solely upon the desire to maintain a 2:1 ratio in the number of subjects in the first 2 groups. An even distribution by race and sex was maintained in the various groups.

The control group was fed a formula containing evaporated or homogenized whole cows' milk in addition to water and carbohydrate according to the "Michigan" plan of infant feeding (55 calories per pound weight; 1 ounce of carbohydrate supplying 120 calories and sufficient water to supply 2 ounces per pound body weight).

The methionine content of whole milk is generally given at about 99 mg. per 100 cc. Evaporated milk[7] is made by removing about 60 per cent of the water from fresh whole milk, and thereby reducing the volume by approximately one-half. This then makes the concentration of all nutrients in undiluted evaporated milk, such as the protein and amino acids of which they are composed, double those found in an equal quantity of whole milk. On this basis the methionine content of undiluted evaporated milk would be on the order of 198 mg. per 100 cc. (99 x 2).

Accordingly, when either whole milk or evaporated milk formulas are fed at the recommended dilutions, the protein content is great enough to supply this amino acid well above the infants' needs except during critical times when optimum nutrition is of the utmost importance, namely, during convalescence from the illnesses and traumata incidental to this period of life. At these times, the protein level of the formula may be at a relatively low level and the danger of methionine deficiency becomes a fact.

According to the results of Albanese et al[8], the optimal methionine requirement of infants is of the order of 65 to 85 mg. per kg. per day, which would be approximately 750 mg. for a 10 kg. infant. Therefore, for the first three months the average infant receives daily 350 to 400 mg. methionine, which, for normal

nutritional demands, excluding critical emergencies, should prove quite adequate. These figures are consistent with the daily dosage of 3 to 6 gm. methionine ordinarily prescribed for a 70 kg. adult as a supplement to a high protein diet.

3. *Diet—Experimental Group.* The experimental group received a similar formula in addition to supplementary methionine in 90 mg., 180 mg., and 360 mg. doses, respectively. This produced respectively a 25 per cent, 50 per cent and 100 per cent increase in dietary methionine content. All of the mothers were instructed to feed the infants as much as could be taken without urging. The daily intake was measured by the mother and reported to the examining physician at each visit.

All the infants of the four groups received equal, and according to current standards, adequate quantities of vitamins A, D and C. At two months of age the infants were given cereal twice daily; at three months vegetables were begun; at four months, egg yolk and fruit.

Observations were made at four-week intervals until six months of age. The two groups receiving 50 per cent and 100 per cent dl-methionine supplement, respectively, were observed for a one-month period (28 days) beginning at three months of age. All clinical examinations upon which this study is based were performed by the same investigator. All infants were weighed to the nearest 10 grams.

CLINICAL OBSERVATIONS

The methionine-supplemented formula was given to 50 infants in 90 mg. doses, to 20 infants in 180 mg. doses, and to 10 infants in 360 mg. doses. In the first group, receiving 90 mg. of supplementary methionine daily, the formula was taken avidly. This was also true for the second group, receiving 180 mg. of supplementary methionine daily. In the third group, receiving 360 mg. of supplementary dl-methionine daily, two of the ten infants did not take all of their prescribed formula, most likely because large quantities of methionine impart a decidedly disagreeable flavor and aroma to the milk. In no instance was there vomiting or diarrhea directly ascribable to the supplementary methionine.

ANALYSIS OF RESULTS

The average gain in weight for the first six months of life of 109 infants used as controls was 4,878 grams or approximately of the order of 813 grams per month (Fig. 1).

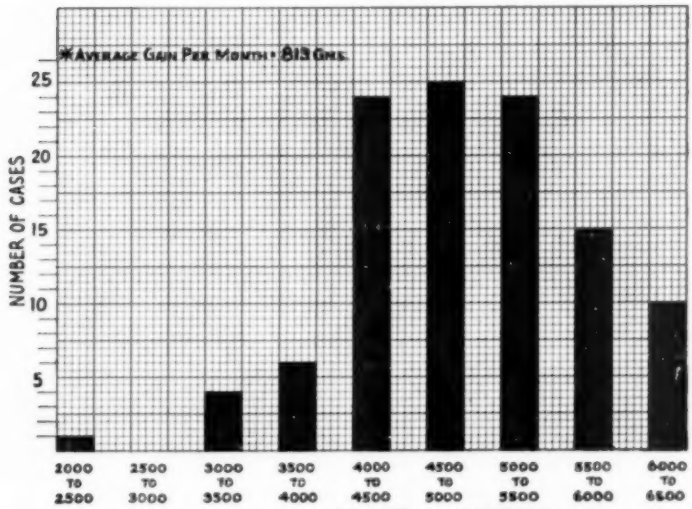

FIGURE I

CONTROLS – GAIN IN WEIGHT FOR FIRST SIX MONTHS OF LIFE.

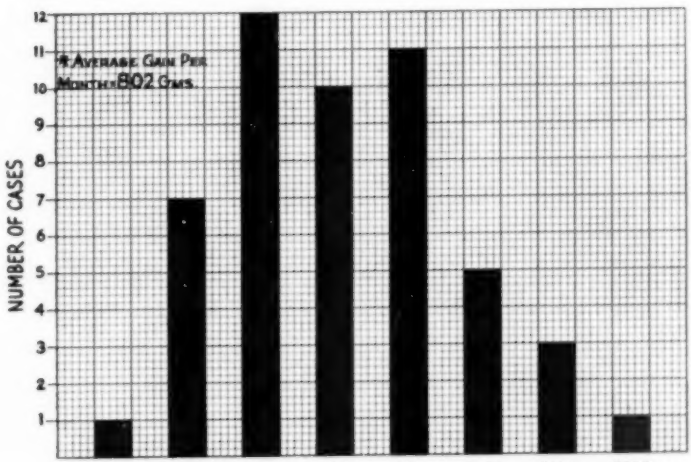

FIGURE II

90 MGS. SUPPLEMENTARY DL-METHIONINE DAILY - GAIN IN WEIGHT FOR FIRST SIX MONTHS OF LIFE —

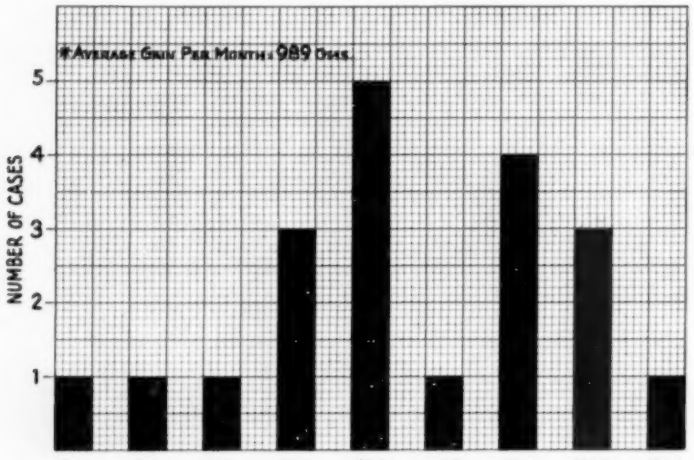

FIGURE III 180 MGS. SUPPLEMENTARY DL-METHIONINE DAILY-GAIN IN WEIGHT FROM THIRD TO FOURTH MONTH OF LIFE (28 DAYS-EXPERIMENTAL PERIOD)

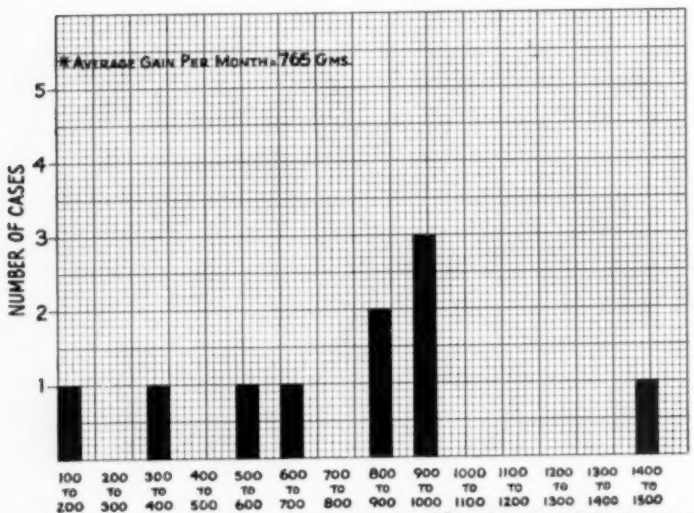

FIGURE IV 360 MGS. SUPPLEMENTARY DL-METHIONINE DAILY-GAIN IN WEIGHT FROM THIRD TO FOURTH MONTH OF LIFE (28 DAYS-EXPERIMENTAL PERIOD)

Fifty infants received a supplement of 90 mg. of dl-methionine daily. This is equivalent to a 25 per cent dietary increase of dl-methionine. During the first six months of life the average gain in weight was 4,813 grams or of the order of 802 grams per month. This approximates the weight gain of the controls (Fig. II).

Twenty infants received a supplement of 180 mg. dl-methionine for twenty-eight days. This is equivalent to a 50 per cent dietary increase of dl-methionine daily. Their average gain in weight was 989 grams per month, an apparent increase of 176 to 187 grams above that of the control and 25 per cent methionine-supplemented groups. This added increment of weight in individuals at this early period of life made possible by supplementary methionine becomes of great interest as well as academic importance (Fig. III).

There were ten infants in the group who received 360 mg. dl-methionine supplement daily for twenty-eight days. This is equivalent to a 100 per cent dietary increase of dl-methionine daily. Their average gain in weight was 765 grams, a figure somewhat below that of the controls as well as the 25 per cent supplemented group. One infant gained only 85 grams and a second infant only 340 grams. At the other extreme, one infant gained 1474 grams. Five of the group fell within the average weight gain of the controls (Fig. IV).

The greatest gain in weight was in the group of infants receiving 180 mg. supplementary methionine (50 per cent) daily. Those receiving 360 mg. showed an apparent impairment in growth but in no instance was there an obvious loss of weight. In terms of percentages, as in the animal experimental groups[4, 5], a 50 per cent supplementary increase in dietary methionine is advantageous and produces an accelerated weight gain, whereas a 100 per cent supplementary increase in dietary methionine definitely produces impaired growth.

DISCUSSION

Through continuous replacement of their constituents the cellular structure of the tissues is preserved. In the growing phase (infancy), the rate of replacement exceeds wear and tear; in the resting state (older animals), replacement keeps pace with wear and tear[9]. It is of primary importance for optimum nutrition that the infant diet be so constructed that the various nutrients be ample and proper for energy, maintenance and growth needs.

If part of the dietary methionine is utilized to furnish methyl groups for detoxication of intermediate metabolites or methylation, for example, of nicotinamide to its methyl derivative, insufficient essential amino acid (methionine) will remain to form complete proteins[10]. This will retard tissue anabolism, inhibit growth, and increase nitrogen excretion. We now know that all the essential amino acids, to be fully effective for tissue growth and maintenance in animals, must be consumed at about the same time and in proper relation to one another[11]. Maximum conversion requires an optimum balance of amino acids. In other words, unless a tissue protein can be synthesized at once and completely, it may not be synthesized at all.

The body does not store amino acids and is, therefore, more or less dependent upon its daily intake for sustained maintenance and growth. The efficiency of any protein depends on the minimum amount of indispensable amino acid it yields[12]. Supplementary methionine provides an excess of a very versatile essential amino acid which promotes completion of the amino acid mixture in the metabolic pool, accelerates nitrogen retention and growth, and in turn diminishes nitrogen excretion. Supplementary methionine, therefore, contributes to a high protein diet. The greater the quantity of indispensable amino acid, the greater the efficiency of the protein up to an optimum beyond which there results a tremendous excess of non-utilizable amino acids which must be excreted. This promotes diuresis and loss of weight[13].

The formation of enzyme complexes is closely dependent on the adequacy of protein nutrition. Since enzymes are the mediators of metabolism, and since the presence of methionine determines the concentration and activity of xanthine oxidase of the liver, an optimum amount of dietary methionine is of paramount importance for maximum growth[14].

SAFETY FACTOR

It is unlikely that any infant will accidentally or voluntarily receive an unusually large, toxic quantity of methionine. Large quantities of supplementary methionine impart a disagreeable flavor and offensive aroma to the milk which the infant will not readily accept. In addition, the conspicuous odor of methionine in the urine as voided would soon become apparent to the most inexperienced mother. Besides under ordinary circumstances methionine

is prescribed only for a few days to eradicate ammoniacal urine, no great harm could ever be done to the infant.

SUMMARY AND CONCLUSIONS

Methionine is a versatile amino acid which plays a strategic rôle in the overall metabolism, participating in fat, protein, sulfur, and water metabolism. A 50 per cent supplement of methionine produces an optimum gain in weight in infants, whereas a 100 per cent supplement obviously impairs growth and produces diuresis. The disagreeable flavor of methionine imparted to the milk, the characteristic odor of the voided urine, and the limited period of use produce a safety factor which insures freedom from ingestion of toxic quantities.

I am under obligation to Lobica-Debruille, Inc., who furnished sufficient Metiogran (containing 10% dl-methionine) for these observations.

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435 *South Broadway.*

OESOPHAGEAL ATRESIA WITH CONGENITAL CARDIAC ANOMALY*

M. A. Abboud, M.R.C.P., D.C.H.
AND
O. Alfy, D.Ch. (Cairo)
Cairo, Egypt.

Congenital oesophageal atresia was formerly regarded as a condition of great rarity, but it is now known to be relatively common. The incidence is stated to be about one in every 2,500 births.

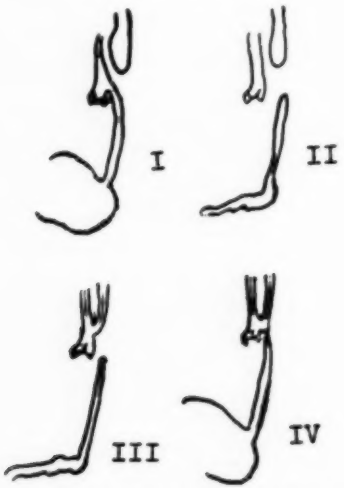

Fig. 1 Showing the various pathological types of oesophageal atresia. Type I is the commonest (70-80 per cent).

There are various pathological types, but the commonest anomaly (70-80 per cent) is for the upper segment to end blindly in a bulbous pouch at the thoracic inlet and the lower segment—usually narrow and poorly developed—to open into the posterior wall of the trachea by a narrow, vertical slit just above the carina (Fig. 1). The other types are rare. There may be other associated congenital abnormalties, such as atresia of the anus or the small intestines or cardiac abnormalties.

Diagnosis should, if possible, be made before any food has been

*From the Paediatric Department, Kasr El-Ainy Hospital, Cairo, Egypt.

given to the infant. Once suspected, the diagnosis can be quickly confirmed by passing a rubber catheter into the upper oesophagus when it will be arrested 3 to 3.5 inches from the alveolar margin. Half a ml. of lipiodol may be injected into the catheter to demonstrate the limits of the upper pouch, but barium should not be used for this purpose.

The case to be recorded is of interest because of its association with a rare type of cardiac anomaly.

CASE REPORT

S. A. H., a full term male infant, was admitted to hospital on the fourth day of life, for attacks of choking, cyanosis and almost

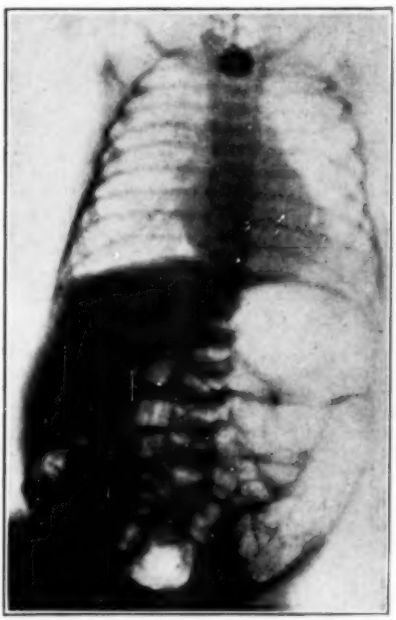

Fig. 2. Showing the arrest of lipiodol in the upper oesophageal segment, and the air distending the stomach and intestines.

immediate regurgitation on being fed. Even as little as a teaspoonful of fluid evoked these attacks. Meconium was passed on the first and second days of life. On examination, the infant was dehydrated, with a markedly and uniformly distended tympanitic

abdomen. He was dyspneic, but not cyanosed. Numerous moist râles were heard over the chest. Large amounts of frothy saliva drooled from its mouth. Excessive amounts of flatus were being passed.

A rubber catheter passed through the nose was arrested 10 cms. from the external nares. Radiography, after injecting ¼ ml. of lipiodol through the catheter, showed the arrest of lipiodol in the

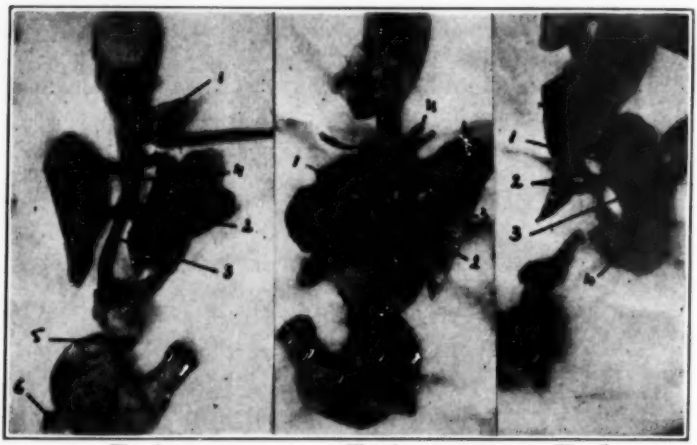

Fig. 3. Fig. 4. Fig. 5.

Fig. 3. A posterior view of the oesophagus showing (1) the upper oesophageal segment exteriorized in the cervical region; (2) the bifurcation of the trachea; (3) the lower oesophageal segment; (4) the fistulous communication between the lower oesophageal segment and the trachea just above the carina; (5) the cardia ligated, and (6) the gastrostomy tube.

Fig. 4. A front view of the heart and great vessels showing (1) the aorta; (2) the pulmonary artery; (3) the left pulmonary artery; (4) the site of coarctation; (5) the patent ductus arteriosus, and (6) the descending aorta.

Fig. 5. A view of the right side of the heart showing (1) superior vena cava; (2) the right pulmonary veins draining into the right auricle; (3) the right auricle, and (4) the right ventricle.

oesophagus at the level of the first dorsal vertebra (Fig. 2). No lipiodol passed to the trachea. Radiography also revealed the excessive amounts of gas in the intestines and evidence of aspiration pneumonia in the chest. Intestinal atresia, which may occasionally be associated, was excluded because of the continuous passage of flatus.

The general condition of the infant was too bad to permit a transthoracic anastomosis of the two oesophageal segments. Instead, gastrostomy and ligation of the cardia were done, followed

the next day by cervical oesophagostomy for the upper blind pouch. Both operations were done under local anaesthesia. The infant died on the third postoperative day.

Autopsy revealed an oesophageal atresia with a blind upper pouch and a fistula communicating the lower segment of the oesophagus to the trachea just about the bifurcation (Fig. 3). The lungs showed evidence of aspiration pneumonia.

There was an associated anomaly of the heart and great vessels: An infantile coarctation of the aorta, with a wide patent ductus arteriosus was present; the aorta regaining its original diameter just beyond the site of the ductus (Fig. 4). The right pulmonary veins were draining into the right auricle (Fig. 5). The left subclavian artery was doubled.

COMMENT

The diagnosis of oesophageal atresia is usually easy provided the condition is borne in mind. The passage of a catheter, oesophagoscopy or radiological examination after the administration of a contrast medium readily establishes the diagnosis; the presence or absence of gas in the stomach points to the existence or otherwise of a tracheo-oesophageal fistula. Barium, as a contrast medium, should not be used to avoid its being aspirated in the lungs. Success in treating this otherwise fatal condition depends on early diagnosis and treatment before dehydration, inanition and/or aspiration pneumonia lead to irreversible changes. The second day of life is considered to be the most suitable time for surgical intervention, and it is surprising how well the infant stands the operation at such an early age.

The associated cardiac lesion in our case was not suspected during life for many reasons: no murmurs were heard, and even, if present, they are usually of no significance at this age. Death occurred too soon for enlargement of the heart or changes to occur in configuration. Any cyanosis present could be attributed to the oesophageal atresia. The difference in cyanosis between the upper and lower limbs which characterizes infantile coarctation was much lessened by the fact that the right pulmonary veins were draining into the right auricle carrying to it oxygenated blood, so making the blood pass to the pulmonary artery, and then through the ductus arteriosus to the lower half of the body, less venous than is usual for cases of infantile coarctation.

PHENYLPYRUVIC OLIGOPHRENIA

HAROLD E. STADLER, M.D.
AND
ROBERT L. DRYER, Ph.D.
Indianapolis.

We are using an isolated instance of phenylpyruvic oligophrenia for the purpose of reviewing the diagnostic features of the disease, and also to clarify the use of certain biochemical procedures necessary to confirm the presence of the disorder.

R. K., female, was born on March 26, 1952. The father and mother of the infant are both healthy and of normal intelligence. One female sibling, 2½ years of age, is normal except for being 5 pounds underweight. R. K. weighed 7 pounds at birth and was apparently normal until the age of four months though head measurements were not made. It was noted, however, that she failed to grasp objects, was very slow in the development of motor activity. There had been no illness other than mild respiratory infections.

The patient was 11 months of age when first observed by the present writers. Her length was 29 inches, the weight was 18½ pounds, and the occipitofrontal head measurement was 39 centimeters. The anterior fontanel was closed. All of the deep reflexes were hyperactive. Moderate rigidity of the back and of the extremities was noted to be present. The head was supported poorly.

X-ray of the skull showed all measurements to be small with complete fusion of all the sutures. The red blood count was 4.23 cells and the white blood count 8,000 cells for each cmm. of blood. The hemoglobin was 75 per cent of normal. The differential smear and routine urinalysis were normal. Three different samples of urine were tested for the presence of phenylpyruvic acid and all three samples gave positive tests with the ferric chloride according to technique of Jervis[1]; in addition, a quantitative assay was carried out according to the procedure of Penrose and Quastel[2]. A level of 0.12 grams of phenylpyruvic acid per 100 ml. of urine was found. No tests were carried out on the

Assistant Professor of Biochemistry, Indiana University School of Medicine, Indianapolis, Indiana (Dryer).

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blood or serum. The ferric chloride test applied to urine samples from both parents and the patient's sibling gave negative results.

DISCUSSION

Jervis[1] has pointed out that the clinical features of phenylpyruvic oligophrenia are intellectual defect, rigid posture, muscular hypertonicity and hyperactivity of the deep reflexes. These cardinal signs were certainly present in our patient. The factor which will lead to the investigation of the patient and the establishment of the diagnosis must in all instances be the awareness of this comparatively rare disease when dealing with mental retardation, particularly when associated with microcephaly. It is of paramount importance to recall that the affected individual constantly excretes phenylpyruvic acid in greater or lesser amounts, but in all cases the acid is detectable in the urine. Normally this substance is not found in the urine since it is an intermediate derivative of the metabolism of phenylalanine which is broken down to water and carbon dioxide. It is stated by Jervis et al.[2] that the primary chemical defect in phenylpyruvic oligophrenia is the inability to dispose of this acid at an adequate rate. Phenylpyruvic acid, which is found in the urine, is presumed to be formed by renal deamination of a hydroxylated phenylalanine.

Several procedures[1,2,3] for the determination of phenylpyruvic acid have been described. These depend respectively upon the formation of a fleeting green color when ferric chloride is added to the urine, or upon the formation of a yellowish precipitate when 2, 4-dinitrophenylhydrazine is added, or finally, upon the production of a mahogany color when the urine is treated with alkaline picrate. Two of these tests are commonly used for the detection of various carbonyl compounds and reducing agents generally and hence may not be absolutely specific. The ferric chloride reagent has been employed for the detection of acetone, but the violet color found in the latter case is persistent. It must be emphasized again that the green color due to phenylpyruvic acid treated with ferric chloride may be so ephemeral as to be missed where the concentration of the phenylpyruvic acid is low. *Erroneous conclusions might be drawn if the alkaline picrate test were applied to a urine unless the urine were previously treated with Lloyd's reagent to remove creatinine, a substance common in urine. Further-*

more, glycosuria will also cause false positives for phenylpyruvic acid when the dinitrophenylhydrazine test is used. For the routine test for phenylpyruvic acid we must agree with Jervis that the most reliable test is the one involving the use of ferric chloride. These facts were demonstrated by one of the authors (R.L.D.) when a falsely positive test for phenylpyruvic acid was obtained on the urine of the mother of R. K. by means of the alkaline picrate and the dinitrophenylhydrazine tests. It was determined that this patient had a moderate glycosuria; the final criterion had to be the negative ferric chloride test.

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SURGICAL TREATMENT OF HEART DISEASES IN CHILDREN. (Nordisk Medicin, Stockholm, 47: 383, March 21, 1952). Mannheimer reviews the clinical diagnosis and indications for operative treatment of congenital heart disease in children, and urges that all patients with congenital heart defects be admitted to special cardiological departments for examination. He considers briefly congenital heart defects that do not produce cyanosis, such as patent Botallo's duct, coarctation of aorta, anomalies of aorta and subclavian artery, and pulmonary stenosis without an overriding aorta and without septal defects. He discusses the defects that cause cyanosis, such as the tetralogy of Fallot, pulmonary atresia with overriding aorta and defect of the interventricular septum, tricuspid atresia, and Fallot's triad. Valvulotomy by Brock's method done by Crafoord in 10 cases of pulmonary stenosis with septal defect resulted in improvement in eight cases; two patients died following the intervention. Follow-up examination of 78 cyanotic children, 76 with the tetralogy of Fallot and two with tricuspid atresia, operated on in Stockholm from late 1947 to early 1950, mainly at the age of from 2 to 10, showed 10 deaths. The mortality, however, has been decreasing steadily, and the results of the anastomosis operation in suitable cases of congenital cyanosis are considered encouraging.—*Journal A.M.A.*

MATHEMATICS FOR THE PEDIATRICIAN

S. LEVIN, M.B.
Pediatric Service, Boston City Hospital

"You cannot bring up a baby by mathematics." (Dr. Abraham Jacobi).

Jacobi was right, of course. He was referring particularly to the involved dietary calculations in infant feeding. Yet a nodding acquaintance with elementary arithmetic is of much help in everyday pediatric practice.

"Doctor, my boy is 5½ years old and weighs 44 pounds." The physician is often puzzled by such an opening gambit—not knowing whether to click his tongue in sympathy, chide mother for over-feeding the poor lad or reassure her that this weight is quite normal. His reaction has perforce to wait until he has hastily and sometimes stealthily consulted the height-weight tables at his side.

"Doctor, is 3 foot 11 inches a normal height for a girl of 6 years?"

"My baby is 7 weeks old and only takes 4½ ounces at a feed."

"Do you think baby's head looks too big, doctor?"

"How much should he weigh when he's 6 months old?"

Everyday questions like these can be awkward and embarrassing. The physician must play for time and consult tables before giving his opinion.

This paper is written to help the practitioner and pediatrician overcome some of these difficulties. It is perfectly simple to calculate normal weights and heights in a fraction of a second without recourse to tables. It is merely necessary to remember two or three very simple formulas. Everyday use of these for a week will make them "second nature" to the pediatrician.

Weights. It is commonly taught that infants double their birth-weight at 5-6 months and treble it in a year. Calculations involving this principle are too involved, and in any event are inaccurate in that they tend to give underweight values. It is stressed that all infants are a law unto themselves. A normal mature three-month-old infant may weigh 9 pounds or 16 pounds, but for most mature babies the following scheme is approximately true:

A	baby	at	birth	weighs	?
"	"	"	1 month	"	?
"	"	"	2 months	"	?
"	"	"	3 months	"	13 lbs.
"	"	"	4 months	"	14 lbs.
"	"	"	5 months	"	15 lbs.
"	"	"	6 months	"	16 lbs.
"	"	"	7 months	"	17 lbs.
"	"	"	8 months	"	18 lbs.
"	"	"	9 months	"	19 lbs.
"	"	"	10 months	"	20 lbs.
"	"	"	11 months	"	21 lbs.
"	"	"	12 months	"	22 lbs.

What about children over the age of a year? The following are weights of normal children aged 1-15 years. The sex difference is so minor as to be of no significance. Again, it is stressed that normal weights may vary tremendously, especially after the age of 10 years. The following table gives the *lower limit* normal weight figures, rather than the optimum figures. This has an advantage in that the large number of parents, who think their offspring are underweight, can be reassured in that respect.

Most	children	at	1	year	weigh	20	lbs.	or	more.
"	"	"	2	years	"	25	"	"	"
"	"	"	3	"	"	30	"	"	"
"	"	"	4	"	"	35	"	"	"
"	"	"	5	"	"	40	"	"	"
"	"	"	6	"	"	45	"	"	"
"	"	"	7	"	"	50	"	"	"
"	"	"	8	"	"	55	"	"	"
"	"	"	9	"	"	60	"	"	"
"	"	"	10	"	"	65-70	lbs.	or	more.
"	"	"	11	"	"	80	"	"	"
"	"	"	12	"	"	90	"	"	"
"	"	"	13	"	"	100	"	"	"
"	"	"	14	"	"	110	"	"	"
"	"	"	15	"	"	120	"	"	"

Nowadays, most children weigh in fact some 5 pounds more than shown in the tables; nevertheless, the figures given are normal values, although they tend toward the lower level of normalcy. It may be noted that children gain 5 pounds a year from 1-10 years and 10 pounds a year thereafter. Bearing this in mind there is a simple formula which gives in an instant the normal weight of a child between 1-10 years: *(age in years + 3) × 5 = weight in pounds,* e.g., a child of 7 years should weigh 10 × 5 = 50 pounds. After the age of ten years (when a child should weigh 65-70 pounds) a formula is not needed if it is remembered that 10 pounds is gained every year.

Heights. Again, heights of children vary a great deal, but for most boys and girls the following table holds true:

At	1	year	most	children	are	30 inches
"	2	years	"	"	"	33 "
"	3	"	"	"	"	36 "
"	4	"	"	"	"	39 "
"	5	"	"	"	"	42 "
"	6	"	"	"	"	45 "
"	7	"	"	"	"	48 "
"	8	"	"	"	"	50 "
"	9	"	"	"	"	52 "
"	10	"	"	"	"	54 "
"	11	"	"	"	"	56 "
"	12	"	"	"	"	58 "
"	13	"	"	"	"	60 "
"	14	"	"	"	"	62 "

It is clear that from 1-7 years children grow 3 inches a year, and from 8-14 years, 2 inches a year. This is simple arithmetical progression, so that a formula for calculation of height immediately suggests itself:

a. From 1-7 years: take the age in years after the basic standard of 1 year. Multiply by 3 and add the result to the basic 30 inch height, e.g., normal height for a 5-year-old is (4 x 3) + 30 = 42 inches.

b. From 8-14 years: take the age in years after the basic standard of 8 years. Multiply by 2 and add the result to the basic

50 inches height, e.g., normal height for a 12-year1old is (4 x 2) + 50 = 58 inches.

Skull. There are so few figures to remember in skull measurements that "a formula" would be out of place. Experience teaches pediatricians to remember a few important figures:

Birth	13-14 inches
7 months	17 "
1 year	18 "
5 years	20 "
10 "	21 "
15 "	22 "

Urine. It is seldom necessary to know the total daily urinary output, but occasionally the need arises. A few "key figures" are outputs, but occasionally the need arises. A few "key figures" are all that need be remembered:

At 1 month a child passes up to	10 oz. urine daily
" 1 year " " " "	15-20 oz. " "
" 5 years " " " "	30 oz. " " "
" 10 " " " " "	40 oz. " " "
An adult " " "	50 oz. " " "

Feeding. "My baby is seven weeks old and takes 4½ ounces, Doctor." This poses a question: "Too much; too little?" Certainly babies vary greatly in their fluid intake. A 5-month-old infant may be satisfied with 5 or 6 ounces, or may take up to 12 ounces at a feed, but generally speaking, *an infant takes in ounces what its age is in months +3*, i.e.:

A mature baby at birth	takes 3 oz. at a feed
" " " " 1 month	" 4 " " " "
" " " " 2 months	" 5 " " " "
" " " " 3 "	" 6 " " " "
" " " " 4 "	" 7 " " " "
" " " " 5 "	" 8 " " " "

Generally speaking, infants of 5 months and over are usually satisfied with an 8 ounce feed.

Another easy method of calculating individual feeds is based on weight rather than age: An infant will take in ounces half of what

he weighs in pounds, i.e., a 12-pound infant will usually take about 6 ounces at a feed.

Infectious Fevers. There is no easy method (other than a mnemonic) for remembering incubation periods. These figures are learned by experience. Perhaps more important knowledge relates to the answer of a common question: "How long will he have the fever before the rash appears, Doctor?" This will not stump an experienced pediatrician. Occasionally the tyro has to think twice to answer that one. He need only think once, however, if the following mnemonic is committed to memory:

1.	Really	Rubella	Rash appears 1st day of illness
2.	Very	Varicella	" " 2nd " " "
3.	Sick	Scarlatina	" " 3rd " " "
4.	People	Pox (smallpox)	" " 4th " " "
5.	Must	Measles	" " 5th " " "
6.	Take	Typhus	" " 6th " " "
7.	Ease	Enteric (typhoid)	" " 7th " " "

This mnemonic is inadequate in that a few exanthemata are omitted, e.g., roseola infantum (rash on 3-4th day), infectious mononucleosis (rash on 4-10th day), but by and large it has been found to be very useful in everyday practice in a busy pediatric service.

AUREOMYCIN IN INOCULATION VACCINIA. (Medizinische Welt, Stuttgart, 20: 747, June 2, 1951). A woman, 25, had been subject to eczema since the age of 10. The present disorder began with the appearance of some pustules around the mouth, to which the patient applied boric acid ointment and alcohol. A physician injected calcium and prescribed an ointment, but despite this treatment the skin lesions advanced so rapidly that hospitalization became necessary. Face, neck, and chest were covered with pustules and yellowish crusts, and the face was edematous. So far as the patient knew, she had not been in contact with newly vaccinated children, but the eruption occurred at a time when many children were being revaccinated. This fact, the nature of the cutaneous lesions, and the demonstration of Paschen's inclusion bodies justified the diagnosis of inoculation vaccinia. The cutaneous lesions and fever subsided rapidly with aureomycin therapy.—*Journal A.M.A.*

PEDIATRICS AT THE TURN OF THE CENTURY

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

TREATMENT OF SUMMER DIARRHEA IN CHILDREN —SOME SUGGESTIONS.*

W. L. HARRIS, M.D.
Norfolk, Va.

There has been so much written on the subject of infantile diarrhea within the last few years, I hesitate to attempt to add anything more, but the phase of the subject which I shall briefly consider I hope may prove of some value to those who are interested in the subject.

The class of diseases designated by the term summer diarrhea seems to be universal in warm climates, and has probably existed from earliest times, but not recognized or treated as a specific disease in this country till the latter part of the eighteenth century, when Benjamin Rush wrote a very valuable article on the subject. It was then called cholera infantum and embraced all the serious types of diarheal diseases now included under the term summer diarrhea. These diseases were almost universally known by the term cholera infantum till the latter part of the nineteenth century, and the term is still erroneously used by some, but the majority of physicians use it now only in its proper sense. There have been many attempts at naming and classifying the various types of the diarrheal diseases as seen in summer, but none has proved quite satisfactory, and the term summer diarrhea is now quite generally used, although there are many reasons why it is a misnomer. The reason the term is popular is that although some types of diarrhea are seen in children during the winter, they are seldom fatal or very prevalent except in summer.

There have been varoius theories as to the cause of these diseases, chief of which have been teething, heat and milk poisoning, but, in the light of etiology and pathology of other diseases,

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in recent years these diseases have been thought to be of bacterial origin. Booker, of Baltimore, was the first man in this country who did any work of great value towards solving the question of etiology. After Shiga's discovery of the bacillus that caused dysentery in adults in Japan, and Flexner's work along the same line in Manila, Flexner took up the subject with reference to establishing this same organism as the cause of the diarrheas of infancy. Working under a grant of money from the Rockerfeller Institute of Medical Research, Dr. Flexner and his various associates, notably Drs. Duvall and Bassett, have done a great deal towards establishing a positive causal relation between the bacillus dysenteriae and the summer diarrheas of children. The new clue as to etiology was greeted with a great deal of interest, and it put renewed life into the work in the hope of being able to find a specific curative serum since a specific cause had been found. Although this subject has been worked upon faithfully by various investigators for the last four years, as yet the results have been disappointing.

There are a great many conditions to be overcome before we may expect satisfactory results from a serum. If it could be proven positively that the bacillus dysenteriæ is the sole cause of the trouble we might soon expect to get better results from a serum; but, as Dr. Holt says, there are so many conditions to be taken into consideration in the treatment of this disease, the outlook is not as bright as it might otherwise be; so much of the child's trouble may be dependent upon its state of digestion, nutrition, etc. If the bacillus dysenteriæ alone was the cause, it seems but reasonable to suppose that in the near future we may expect better results from the serum, especially in children previously in good condition taken suddenly ill with the acute infectious type of the disease.

Laying aside the specific etiology and treatment looked for, let us see what has been accomplished in other ways of treatment. It would undoubtedly be of interest to compare the death rate now with that of former years, but statistics of any value are not obtainable. All the statistics on the subject give only the actual number of deaths, and none of them give percentage of mortality in any given number of cases. I have experienced the greatest difficulty in securing any statistics of value on the subject.

We all know the great majority of cases of diarrhea in summer are simple and mild, and if properly treated will almost invariably recover, but, of course, if allowed to run on and be neglected for any reason, they soon drift into the serious forms of diarrhea. We also know that there is a certain fair proportion of cases that are serious from the start, and as soon as we see them we know they are ill; there is fever which continues, great prostration, frequent actions, green and full of mucus and perhaps tinged with blood; and in spite of everything we can do, these cases continue ill for a certain variable time, from a few days to few weeks, before there is any sign of improvement.

These last cases can readily be put down as infectious, whereas, the first cases were not infectious. In making up statistics it does not give a fair idea of the mortality of summer diarrhea to include all these simple cases, from which we have practically no mortality, with those of a serious type from which we all know we have yet a frightful mortality; yet this is just what is done in almost every instance. It gives an incorrect idea of the seriousness of summer diarrhea. If, for example, you treat 300 cases of so-called summer diarrhea, 200 of these cases are simple and mild and uncomplicated, and, in fact, you could scarcely call the child sick, having only a few loose and perhaps mucous stools, and if properly treated all these cases get well; and then the other 100 cases are of the severe type and your morality is 30 per cent. It would be giving the wrong impression to say your mortality of 300 cases of summer diarrhea is 10 per cent., yet this is practically what is done right along. Of course every man wants his statistics to show up the best possible, but there should be some definite clinical classification of these diseases in compiling statistics to be of any use to us.

In the Wilson Sanitarium near Baltimore, where three or four hundred sick children are sent every summer, Dr. Knox, physician-in-charge, writes that his total mortality is about 15 per cent., but that if he eliminated the simple cases from the list, the mortality in the more serious types of the disease is anywhere from 40 per cent. to 50 per cent. This corresponds very closely to my experience in the Virginia Beach Infant Sanitarium. Of course, it must be taken into consideration that children sent to these institutions are from the poorer classes and many cases are far advanced, and the great majority of them before they were

taken ill were suffering from some form of indigestion and in many cases the children are decidedly marantic.

Dr. Kerley, of New York, in giving his statistics in 682 cases, treated in the out-patient department of several institutions, states that only 21 cases died, a mortality of only a little over 3 per cent. This number of cases includes all varieties of the disease, the simple and mild, as well as severe. It is safe to say that the great majority of these cases must have been simple and mild, which would raise his mortality among the severe type to a much higher figure.

In my experience in dispensary work in this line we have very few of the very ill cases brought into the dispensary (probably not 1 case in 6 or 8 is very sick). I don't know why we see so few of the worst types in the dispensaries; they probably are attended at home by a family physician or city physician, or die before the mothers can make up their minds they are sick enough to take to a physician; so it seems to me that Dr. Kerley's statistics do not convey to us anything like the real seriousness of summer diarrhea as it occurs among the poor; certainly not as it occurs in this section.

Statistics from the Boston Floating Hospital are about an average I should say. During the past summer 279 patients were cared for in the permanent wards of the boat, with 90 deaths, 90 cases discharged well, 71 relieved and 28 not relieved. Of the 90 deaths, 26 occurred within 48 hours after admission. Of the 28 cases not relieved, probably quite a number subsequently died. Not all of the 279 cases were diarrheal cases, but probably about 80 per cent. of them were, so that would give a mortality from this trouble between 28 and 30 per cent.

Dr. Dunn, of Boston, has given us a very excellent paper on the subject of etiology and classification of a large series—620 consecutive cases of summer diarrhea as seen in the out-patient department in one of the Boston dispensaries. Of the 620 cases, he puts down 111 as of the acute infectious class, with rapid onset and persistence of fever. One case not classed as infectious died, and of the cases classed as infectious 9 died, and there was no improvement in 17 others. It would be but natural to suppose that the great majority, if not all, of these unimproved ones subsequently died, so this would raise the mortality to between 15 and 18 per cent.

Of course, it is but natural to expect a high mortality among the class of cases spoken of above, but how often do we see children in the best families, strong and well-nourished, taken suddenly ill with diarrhea, and die in spite of all our present medical skill can do for them. When we see so many of these strong and well-nourished children take sick and die, we have to confess that our treatment at present is far from what it should be, and is very ineffective, and we naturally stop and ask what progress have we made in therapeutics along this line. Undoubtedly our treatment is far more rational than formerly even if it is not as effective as we think it should be. Some of the fundamental principles of therapeutics we have learned from experience in recent years are of great comfort even when not of decided benefit to our little patients. We have stopped giving so many nauseous drugs, and have learned that so few drugs do any good at all in this disease. We have pretty generally come to the conclusion that it is best for the child if we leave off many of the drugs that were formerly used in the hope of antiseptic or astringent action. We now realize that it is not our sole aim to stop or check the diarrhea, but to sustain the patient in every way possible and assist nature to combat the infection. We have found astringents practically useless in the serious types of the disease, and about the only drug that is generally used is bismuth in some form. We have fully learned the importance of trying to keep the child's stomach in good condition and to do all we can to help digestion and promote nutrition till the child is able to overcome the infection. We have learned that persistent feeding and drugging, as formerly practiced, can do great harm, and we have also learned that the problem of selecting a suitable and harmless food is often a very perplexing one. Probably the point of greatest value we have learned in feeding during these acute attacks is to stop all milk at once in the acute cases and not resume it till all acute symptoms have disappeared.

The question of feeding and nutrition in the long-continued serious cases is still very unsatisfactory. Probably the best substitute food now generally used is a dextrinized gruel of either rice, barley or wheat. This is, as a rule, harmless; it may ferment, but it does not putrify, and offers a pure culture medium in the intestines, and besides the child can take a large enough

quantity of it to satisfy him without doing him any harm. The various broths may help us over an emergency for a while, but we cannot give them in large enough quantity to do much good for fear of increasing the diarrhea. Beef juice is satisfactory only to a slight degree and we can give it only in small quantities. Albumen water was formerly much used, but now not so generally; it decomposes and putrifies and is not digested or absorbed in the intestines. Unquestionably our best results in reducing infant mortality from summer diarrhea have been accomplished through prophylactic measures, and our greatest hope in the future lies along this line. This disease is undoubtedly to a great extent preventable, and our knowledge of the bacterial origin of it should widen our field of prophylaxis. The bacillus dysenteriæ has not as yet been isolatd outside of the intestines or intestinal discharges, but from our present knowledge it is fair to suppose that it does exist outside of the body and that it enters the body through the food, most probably through water or milk contaminated by water; as the infection lies in the discharges, this known source should be carefully guarded, but it should be borne in mind that at all times there seem to be bacteria in the intestines capable of doing great harm if only proper conditoins are supplied through indigestion and fermentation and the irritation thereby produced. We know that these diseases are most prevalent in the badly fed and poorly nourished, and more apt to be fatal in this class of cases, so it is clearly our duty to educate the laity in general, and mothers and nurses in particular, in the best methods of infant feeding. Of course, the ideal feeding is maternal nursing, and this is more and more impressed upon me the more I see of artificial feeding, and we should impress this fact upon mothers and we should do everything possible to see that the prospective mother is able to nurse her child when it comes and never give up the breast and resort to artificial feeding until we have done everything possible, and proven to ourselves beyond all doubt that the mother cannot nurse the child. Even if she has milk enough to nurse the child only three or four times a day, insist on her doing this much and fill in the balance of the feedings with properly modified cow's milk. If the child has to be artificially fed we should convince the mother that this is often a difficult problem, especially for the first few months, and that it is of enough im-

portance to require the aid of a physician. In giving instructions to mothers or nurses, instructions should be explicit in writing, and we should enter into every detail, and impress upon them the fact that these instructions should be carried out to the letter, and that she should at all times keep in touch with her physician, and that if the child is not doing as well as she thinks it should, she should at once notify her physician. The child should always be weighed at birth and the weight recorded. Select a food the child can digest and assimilate. If the child shows signs of indigestion, note carefully the condition and see what is wrong and make a change of percentage of the ingredients at once. Have the child weighed at regular and stated intervals and see that it gains steadily in weight. If it does not gain steadily in weight it is either not getting enough food or is getting what it cannot properly digest and assimilate. A loss or standstill in weight after the first week demands immediate attention. Do not delay until the digestive tract is impaired, for a slight trouble that is simple at first, and can easily be corrected, may take months to overcome if allowed to run on too long.

Of course, fresh, clean milk should be used in feeding infants, but it is not alone clean milk that has reduced and will reduce infant mortality, but good care and attention in every sense of the word.

Besides instructing mothers how to feed children properly, we should instruct them that at the first approach of hot weather in the spring they should be on the lookout for any intestinal disturbances, and if the child shows any sign of indigestion, should dilute the food more, and that at the first signs of diarrhea to stop all milk at once, give a purgative and feed the child on barley or rice water until she can notify her physician, which should be at once, if possible, for there is no type of diarrhea, it matters not how simple, that is not serious enough to demand prompt attention and treatment in hot weather. If mothers fully realized this fact and could only disabuse their minds of the idea that every diarrhea in summer was due to the natural and benign cause of teething, we should have fewer of the more serious types of diarrhea and fewer deaths to record among children every summer. It is indeed strange how this old idea of teething clings to mothers from one generation to another. It is little less than criminal in many cases the way mothers persist in diagnosing and treating

their children in summer, under the delusion that teething is causing all the trouble. During the past summer a mother who had been in great distress over her sick child that had been desperately ill for three weeks said to me one day: "Doctor, I still believe it is teething," and again in a few days after this she said: "If that other tooth would only come through I think he would get well."

In closing, I will venture the statement that, notwithstanding all that has been accomplished in the matter of infant feeding and prevention of summer diarrhea, if every physician fully realized the importance of what can be done toward preventing this disease, and would only take the trouble in every case that comes under his care to put his knowledge into practice, we could reduce the mortality from this disease probably 50 per cent within a comparatively few years.

DECREASING MORTALITY FROM WHOOPING COUGH IN NURSING INFANTS. (Nederlandsch Tijdschrift v. Ceneeskunde, Amsterdam, 96: 598, March 15, 1952.) Minkenhof and ven der Kooi gained the impression that during the last 50 years infant mortality from whooping cough has decreased not only as the result of more spontaneous recoveries but also because of more effective treatment of secondary pneumonia. Furthermore chloramphenicol and aureomycin do not influence uncomplicated whooping cough favorably. On the contrary, they found that in some cases these drugs render other bacteria, such as those causing pyoderma, resistant to all chemotherapeutic and antibiotic drugs. However, secondary broncho-pneumonia that has proved resistant to sulfonamides, penicillin, and streptomycin responds favorably to these drugs. Investigating the deaths among 249 infants with whooping cough who had not been immunized against this disease, the authors found that while in 1946 there were 22 fatalities among 56 cases, there were none among 40 in 1951. Secondary pneumonia was treated with sulfonamides or dihydrostreptomycin. Aureomycin was used and proved effective in only two resistant cases. Since 1948, infants with whooping cough have been given dihydrostreptomycin in order to prevent pneumonia.—*Journal A.M.A.*

DEPARTMENT OF ABSTRACTS

FRANKLIN, J. E.: ANALYSIS OF CANCER IN INFANCY AND CHILDHOOD. (New York State Journal of Medicine, 53:1659, July 15, 1953).

Neuroblastoma is one of the most frequently seen malignant tumors in childhood. It is usually a silent tumor prior to metastases which makes its early diagnosis most difficult. Wilms' tumor is the second commonest malignant tumor in childhood and is the most common renal tumor in the juvenile age group. The majority of these tumors appear during the first three years of life. The signs and symptoms are invariably absent during the early growth and its presence is detected only by palpation of the abdomen. Lymphosarcoma is a malignant neoplasm arising from lymphoid tissue. The clinical sites of greatest frequency in children were the neck, abdomen, retroperitoneal regions and the bones. The commonest complaint is swelling, which may be in almost any part of the body, but it is not unusual to have obstructive symptoms of the gastrointestinal tract or disturbances of the central nervous system. Retinoblastoma is a congenital malignant tumor representing immature developing retinal tissue. It usually occurs in infancy and childhood with an occasional case being found at birth and an occasional one after 12 years of age. These are diagnosed when they have grown sufficiently to give a whitish appearance to the pupil. At times strabismus or unequal pupils may be the first sign. Osteogenic sarcoma is the commonest malignant tumor arising primarily from bone. It is usually found between 10-20 years of age. Pain is the earliest and commonest symptom occurring in the affected bone, starting as a transitory feature and increasing rapidly in severity with marked loss of sleep. Early metastasis to the lungs is common. Ewing's sarcoma is a relatively rare malignant bone tumor usually arising in the medullary portion of long and flat bones. Pain, swelling and disability of the affected part are the common symptoms. It metastasizes most commonly to the lungs, bones and lymph nodes. Teratomas are embryonal neoplasms composed of multiple tissues foreign to the part where the tumor is found. They are usually present at birth, but some remain quiescent during childhood. The commonest sites or origins are ovary, testis, epididymis, retroperitoneal, presacral and coccygeal,

mediastinal, intrapericardial and brain, in that order. Rhabdomyosarcomas are embryonic growth derived from immature myoblastic or undifferentiated mesenchymal tissue. They are rapidly growing tumors that tend to spread early. The chief symptom and complaint in most of these cases has been a swelling.

MICHAEL A. BRESCIA, M.D

HAMMON, W. MC D.: LIMITATIONS IN THE USE OF GAMMA GLOBULIN IN POLIOMYELITIS. (American Journal of Medical Sciences, *226*:125, Aug. 1953).

There are serious limitations in the use of gamma globulin in poliomyelitis prophylaxis. These are due in part to the fact that this is an agent giving temporary, passive protection only. However, still more important are the difficulties presented by the disease itself; a majority of infections unrecognized, exposure unrecognized in time or place, and susceptibles indistinguishable from immunes. This leaves no alternative to shotgun use of any immunizing agent, gamma globulin or vaccine, but one should aim over a shutgun, when possible, to increase the ratio of kills per shots or pellets used. AUTHOR'S SUMMARY.

SIEGEL, M. AND GREENBERG, M.: PASSIVE IMMUNIZATION IN RELATION TO MULTIPLE CASES OF POLIOMYELITIS IN THE HOUSEHOLD. EPIDEMIOLOGIC ASPECTS IN NEW YORK CITY, 1949-1952. (New England Journal of Medicine, 249:171, July 30, 1953).

The epidemiology of multiple household infections occurring in New York City from 1949 to 1952 was studied with particular reference to the applicability of passive immunization as a community control measure. The percentage of households affected with more than one case of poliomyelitis in a given year varied with the annual incidence of 2.4 to 4.4. The incidence of multiple household infections was related to the number of household members under 18 years of age and to the annual attack rate in the general population. The time interval between the onset of the initial and subsequent cases in a household followed a characteristic pattern, most of the cases occurring within the first week, about 95 per cent within 2 weeks, and 99 to 100 per cent within 5 weeks. The percentage of cases after the first week seemed to be somewhat higher among persons 15 to 34 than among those under 15 years

of age. The incidence of poliomyelitis in household contacts after the onset of the index case was about 40 times greater than that of the general population. It was proportionately greater at 15 to 34 than below 14 years of age. In the group from 15 to 34, the frequency of bulbar poliomyelitis and the mortality were also increased. AUTHORS' SUMMARY.

STEWART, A. AND WESTROPP, C.: BREAST-FEEDING IN THE OXFORD CHILD HEALTH SURVEY. PART II. COMPARISON OF BOTTLE- AND BREAST-FED BABIES. (British Medical Journal, 4831:305 Aug. 8, 1953).

The early progress of 580 babies in the Oxford Child Health Survey has been studied in relation to feeding habits. At the age of 1 year the babies who were bottle-fed from the beginning were appreciably heavier than the babies who were completely breast-fed for 5 months or longer. They also showed signs of being slightly more mature. It is suggested that these differences are due to minor degrees of under-feeding among the breast-fed babies. For 3 disease groups—gastro-enteritis, bronchitis and bronchopneumonia, and otitis media and tonsillitis—there was no evidence that the duration of complete breast-feeding influenced the overall infection rates. Gastro-enteritis was comparatively rare so long as the babies were receiving nothing but breast milk, but it reached its maximum incidence in the weaning period and was especially common among babies who began to be weaned in the sixth month. Colds, which were most frequent in large households and households with several children of school age, were almost equally common among breast- and bottle-fed babies.

AUTHORS' SUMMARY.

MALM, E. AND TALLQUIST, H.: EXPERIMENTS ON RECTAL ADMINISTRATION OF OXYGEN TO NEWBORN ANOXIC KITTENS. (Acta Paediatrica, 42:313, July 1953).

The absorption of oxygen administered into the intestine of newborn anoxic kittens has been investigated. The results indicate that the oxygen content of the blood of markedly anoxic newborn kittens increases during rectal administration of oxygen.

AUTHORS' SUMMARY.

RANSTROM, S.: ON THE EFFECT OF THE HYALINE MEMBRANES IN THE LUNGS OF NEWBORN INFANTS. (Acta Paediatrica, 42:323, July 1953).

An analysis of 30 cases of newborn infants with hyaline membranes in the lungs has led to the conclusion that these membranes are of no significant functional importance in the vast majority of cases. In more than half of the cases death was considered due to pneumonia. In cases where there was no pneumonia or demonstrable extrapulmonary cause of death, the significant factor in the prevention of the gaseous exchange in the lungs was primary and/or secondary atelectasis. The atelectasis was not only located in parts of the lungs corresponding to the membranes. The clinical diagnosis of "membrane asphyxia" in the sense of "asphyxia caused by membrane formation" must be limited to cases with very abundant and widespread membranes causing secondary atelectasis. AUTHOR'S SUMMARY.

KING, J. D.; PATTERSON, J. H. AND BRYAN, W. W.: PREMATURE CLOSURE OF THE CRANIAL SUTURES. (Southern Medical Journal, 46:852, Sept. 1953).

Craniosynostosis may lead to restriction of brain development, skull and facial deformity, convulsions and frequently blindness. These sequelae can often be prevented by early diagnosis and adequate surgical treatment. Such conditions as microcephaly and deformities of the head produced by prolonged dependent positions must not be confused with craniosynostosis. The operative results, particularly in the younger age groups, should be rechecked by x-ray examination every 3-4 months during the first and second year. MICHAEL A. BRESCIA, M.D.

GEFFEN, D. H. AND TRACY, S.: POLIOMYELITIS IN CHILDREN UNDER SIX MONTHS IN ENGLAND AND WALES DURING 1950. (British Medical Journal, 4833:427, Aug. 22, 1953).

A report is presented of 82 cases of poliomyelitis in children under 6 months which occurred in England and Wales during 1950. Of the 82 cases, 77 were paralytic and 5 non-paralytic. The incidence of paralytic poliomyelitis in infants under 6 months was 56.6 per cent of what it would have been if such incidence had

been equally distributed in 6-months age groups in children under 15 years. Non-paralytic poliomyelitis is frequently diagnosed in children under 6 months. The overall mortality rate was 23 per cent, and this mortality increased as age diminished. In children under 9 weeks the mortality rate was 37.5 per cent. Of the 58 paralytic cases who survived, 30 were very severely paralyzed, and of these only 2 made a complete recovery. The pre-paralytic symptoms were not diagnostic except of an acute infection in a child under 6 months. MICHAEL A. BRESCIA, M.D.

HILL, L. W.: ORAL CORTISONE TREATMENT OF INFANTILE ECZEMA. (New England Journal of Medicine, 248:1051, June 18, 1953).

Eighteen eczematous children, ranging in age from 4½ months to 3 years of age, were treated with orally administered cortisone. The duration of treatment varied between 1½ and 11 months, the average being 3 to 6 months. The results of treatment were superior in 8 cases, worth-while in 6 and unsatisfactory in 4. The only immediate adverse side effects were the production of Cushing's syndrome in 1 patient and potassium deficiency in another, when the dose was too large (150 mg.) Cortisone given orally is a valuable addition to local and immunologic treatment for some infants with severe eczema. It is possible to continue cortisone treatment for several months without apparent harm to the child. The dosage should be kept as small as possible; except for extremely critical situations it is probably best not to use over 75 mg. a day. No evidence can be offered, however, that it might not be possible to use somewhat larger doses with advantage. Treatment with cortisone is symptomatic only, for the purpose of tiding the child over a difficult period, and during treatment dietetic and environmental control and local therapy must not be neglected. Although no harm is apparent in most infants treated for relatively long periods with modern doses of cortisone, this treatment should be used for severe cases only, and for as short a time as possible, to relieve an immediate, distressing situation. An entire cure is not expected, and it is better to maintain the eczema under reasonably good control with moderate doses than to suppress it entirely with large doses. AUTHORS' SUMMARY.

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